STUDY OF BLOOD GLUCOSE CHANGES DURING GENERAL ANAESTHESIA IN PATIENTS UNDERGOING SURGERY

THESIS

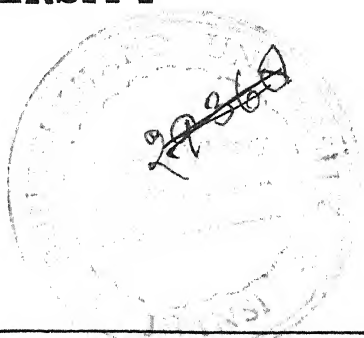
FOR

DOCTOR OF MEDICINE

(ANAESTHESIOLOGY)

BUNDELKHAND UNIVERSITY

JHANSI (U. P.)



1993

HARI NATH KUMAR

C O N T E N T S

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C E R T I F I C A T E

Certified that the work entitled "STUDY OF BLOOD GLUCOSE CHANGES DURING GENERAL ANAESTHESIA IN PATIENTS UNDERGOING SURGERY" which is being submitted as a thesis for M.D. (Anaesthesiology) was conducted by Dr. HARI NATH KUMAR himself in the department of Anaesthesiology, M.L.B. Medical College, Jhansi.

The candidate has fulfilled the necessary stay in the department according to the regulations of the university.

Date: 30.1.92

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C E R T I F I C A T E

This is to certify that the work pertaining to "STUDY OF BLOOD GLUCOSE CHANGES DURING GENERAL ANAESTHESIA IN PATIENTS UNDER GOING SURGERY" which is being submitted as a thesis for M.D. Anaesthesiology by Dr. HARI NATH KUMAR has been carried out under my direct supervision and guidance in the department of Anaesthesiology.

The techniques and methods described were undertaken by the candidate himself and the observations recorded have been periodically checked by me.

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C E R T I F I C A T E

This is to certify that the work of Dr. HARI NATH KUMAR entitled "STUDY OF BLOOD GLUCOSE CHANGES DURING GENERAL ANAESTHESIA IN PATIENTS UNDER GOING SURGERY" which is being submitted as a thesis for M.D. (Anaesthesiology) has been carried out by him in this department under my constant supervision and guidance. The results and observations were checked and varified by me from time to time.

Date: 30 NOV 1992

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A C K N O W L E D G E M E N T S

Words in fact can not substitute for the feeling of gratitude, I have towords all who have helped me in this work.

There can be no greater opportunity than this to express my profound sense of gratitude to my esteemed and exalted guide and teacher Dr. A.K. Gurwara, M.S., D.A., Associate Professor in Department of Anaesthesiology, M.L.B. Medical College, Jhansi, who with his unfathomed knowledge and untiring zest for work guided me unflinchingly throughout this work. The present work has been possible because of his wise counsel, firm initiatives timely and constructive criticism and valuable advice, which I have had the good fortune to receive.

I am highly thankful to Dr. P. Sahi, M.D., D.A., Assistant Professor in Department of Anaesthesiology, M.L.B. Medical College, Jhansi whose valuable guidance and advice in every stage of study, made possible the completion of this work.

I acknowledge with sincere thanks, the affectionate nature, heartening words and constant encouragement of Dr. U.C. Sharma, M.D., D.A., Professor and Head of the Department of Anaesthesiology, M.L.B. Medical College, Jhansi, which has provided me the confidence and enthusiasm so essentially vital for successful accomplishment of such a study.

I am highly obliged and thankfull to all of my senior staff especially Dr. D.D. Verma, M.D., D.A., Assistant Professor of Anaesthesiology and Dr.(Mrs) Veena Gupta, M.D., D.A., Assistant Professor of Anaesthesiology, M.L.B. Medical College, Jhansi, who as perpetual source of inspiration and knowledge bestowed upon me with remarkable generosity and benevolence.

I shall always remain indebted to my father, wife, brothers and sister for their unaccountable sacrifice and their persistant inspiration which enable me to perform this work successfully. I most humbly take the liberty of dedicating this work to my father.

I am thankfull to Mr. B.D. Mathur, Associate Professor in Post Partum Programme, M.L.B. Medical College, Jhansi, who helped me in statistical work and calculations.

I am also thankful to Mr. Zaheer Hasan, clerk, Mr. Om Prakash, Mr. A.P.Singh, O.T. Technicians and Mr. Om Prakash Yadav, ward boy, Department of Anaesthsiology, M.L.B. Medical College, Jhansi, for their co-operation and help time to time.

I am also thankfull to Mr. K.C. Sharma for bringing out the work in representable form by his excellent ability of preparing type scripts.

Lastly I am highly obliged to those patients who become the subjects of this work.

(HARI NATH KUMAR)

Dated: 30.11.92

INTRODUCTION

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Analgesia is meant to reduce the pain produced by surgery. To fulfil this requirement various drugs such as alcohol, opium, hashish and balladona have been used in the past by the Egyptians and Chinese for the control of pain during surgery when the anaesthesia was not known.

Since W.T.G. Morton has given ether inhalation for pain less surgery in 1846 the speciality of anaesthesiology has undergoes tremendous development.

Presently there are various anaesthetic agents. Ether still stands out as the most commonly used anaesthetic agents in our country.

Discovery of muscle relaxant is another mile stone in the development of the smooth and prolonged relaxation during surgery and anaesthesia. The safty of any drug depends on a thorough knowledge and appreciation of its full effects. Much work has been done to evaluate the various drugs for the safty of the patients and their effect on various organ as well as body metabolism As far as ether (inhalation anaesthetic agent) is concerned, the recognition of the injurious effect of ether on the liver and protective effect of high glycogen against these

phenomenon together with Manns observation that anaesthesia causes breakdown of liver glycogen which precepitated in the form of increased level of blood sample due to the effect of general anaesthesia on carbohydrate metabolism.

It is being increasingly realized that several factors related to anaesthesia and operation, such as apprehension, nature of induction, hypoxia and hypercapnoea, and operative stress, might significantly effect the blood sugar level of patients during surgical anaesthesia.

Hence it is natural to have curiosity enough to look into the subject to have enough information as to how much alteration in blood sugar is resulted by using the ether and the muscle relaxant seperatly and have the knowledge of possible cause of such effect.

During this work an attempt has been made to arrive at a conclusion on the mechanism of production of alteration in the blood sugar level by ether and the muscle relaxants when they are used during general anaesthesia for surgery. Having all these facts in the background, observation on blood sugar were carried out at this institution.

At the suggestion and ground plan of Prof. U.C. Sharma Professor and Head of the department of anaesthesiology, M.L.B. Medical College, Jhansi and with the day today guidence of Dr. A.K. Gurwara, Associate Professor and Dr. P. Sahi Assistant Professor, Department of Anaesthesiology, M.L.B. Medical College, Jhansi, an humble attempt has been made by me to observe the effect on blood sugar during general anaesthesia using ether and muscle relaxants (Pancuronium, Vecuronium and Gallamine) with special reference and careful consideration of the fact reported by others.

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MATERIAL AND METHODS

MATERIAL AND METHODS

The present study was conducted in the department of anaesthesiology at M.L.B. Medical College and hospital, Jhansi (U.P.) during year 1991-1992, with the aim to study the blood glucose changes during general anaesthesia in patients undergoing surgery.

Fifty adult indoor patient of either sex between 20-60 years of age scheduled for various elective surgical procedure comprised the material for study.

Patients were devided into four groups of 10 patients each, depending upon the type of anaesthetics used.

Induction of patients of each group was carried out in the same way by thiopentone and suxamethonium for intubation.

The maintenance of anaesthesia was done as follow:-

Group I	O_2 + N_2O + Ether
Group II	O_2 + N_2O + Pancuronium
Group III	O_2 + N_2O + Gallamine
Group IV	O_2 + N_2O + Vecuronium

patients having any respiratory renal hepatic or cardic vascular disease were excluded from the study.

All the patient were of physicaly fit belonging to A.S.A. grade I or II. They were thoroughly examined preoperatively as to their clinical fitness. Routine investigation alongwith relevant special investigation were accepted for the purpose of study.

An informed written consent was obtained from every selected patients and they were kept empty stomach for at least 12 hours before the induction of anaesthesia.

Premedication consisted of injection atropine 0.06mg intramuscularly 30-45 minutes prior to the induction of anaesthesia.

But first blood sample was taken just before the premedication.

Vein puncture was performed with an 16 or 18 gauze I/V canula under proper aseptic condition. Any intravenous drip of Ringer Lactate or Saline was started. Dextrose and Dextrose saline infusion was avoided throughout the study period.

Just before induction the IInd blood sample was taken after recording the pulse, diastolic and systolic blood pressure.

Preoxygenation with 100% oxygen was initiated 3-5 minutes prior to the induction. Induction of anaesthesia was performed with the sleeping dose (4-6mg/kg body weight) of 2.5% thiopentone sodium, slowly till abolition of eye lash reflexes. Intubation was done with proper size cuffed endotrachial tube after injecting suxamethonium in a dose of 1.5 to 2.0mg/kg body weight (80-100mg). The patients were connected to the Boyles Machine using Mapelson 'A' circuit, and ventilated with a gas:oxygen mixture in the ratio of 60:40% respectively, total gas flow being 10 Lts/minute.

Intermittent positive pressure ventilation was continued. IIIrd blood sample was takenout after intubation.

MAINTENANCE OF ANAESTHESIA

Group I (Ether group):- After connecting the patient with Boyle's apparatus anaesthesia was maintained with O_2 and N_2O (40:60) and ether was started and controlled ventilation was contimued until the spontanious respiration resumed IVth blood sample was taken 30 minutes after intubation and adminstration of ether.

Group II (Pancuronium group):- In this group ether was not used. After connecting the patient with Boyle's apparatus positive pressure ventilation was continued. When the effect of suxamethonium were completely then pancuronium was given in the dose of 0.1mg/kg body weight intravenously to keep the patient relaxed and controlled ventilation continued, top up doses were given when required.

Group III (Gallamine triethiodide):- In this group gallamine triethiodide in the dose of 2mg/kg body weight was given intravenously after the effect of suxamethonium was over in the same manner as in case of group II. Controlled ventilation was continued. Top up doses were given when required.

Group IV (Vencuronium):- In this group the patient recieved vecuronium in dose of 0.08mg/kg body weight intravenously in the same manner. Top up dose was given when required.

Supplementation dose of fortwin and phenargan was given to maintain the analgesia during surgery, but hyper or hypo ventilation was avoided.

REVERSAL

Reversal of patient group I was spontanious after the with drawl of the anaesthetic drugs. For the rest of 4 groups the reversal was needed as follows:-

At the end of surgery when there was return of flickering movement in rebreathing bag the residual relaxant were anatogonized with 1.2mg atropine and 2.5mg neostigmine intravenously in all groups except Ist group.

Patients were extubated after establishment of spontaneous respiration. Suction was done for clearing the oral cavity from secretion just before extubation and after extubation. Patients were oxygenated with 100% oxygen for 5 to 10 minutes after extubation. Post operative sample of blood was collected as a fifth sample.

Blood glucose estimation was done with the help of glucometer.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Anaesthesia was not known prior to 1846 although the anaesthetic properties of ether was already discribed by Faraday in 1818.

The use of ether in 1846 by W.T.G. Mortan opened up a new era of painless surgery with the help of drugs (Price and Dripps 1965).

Before this event, chloroform was discovered in 1831 in United States France and Germany simultaniously but was successfully used as a general anaesthetic by James young Simpson in 1841. After that throughout the most part of world it became the anaesthetic agent of choice. But due to outstanding demerits of chloroform anaesthesia, like, fall in blood pressure, cardiac arrest etc. it became less popularized and some workers termed it as a dangerous drug due to its delayed poisoning.

By 1894 ether gained popularity due to the introduction of open ether by Jafferson in 1872 and just after this in 1874 Clover introduce the gas ether sequences. But the open ether technique reaches America in 1895.

When the pure sample of cyclopropane was prepared and tested by Hewer and Hadfield in 1941, it proved to be more potent and suitable anaesthetic agent due to cheap cost noninflamability and little depresent action on respiration blood pressure and cardia output.

With the introduction of newer volatile anaesthetic agent and muscle relaxant popularity of cyclopropane declined.

Krantz and associate (1953) evaluated the anaesthetic properties of trifloroethylvinyl ether and used clinically in 1961. He found that this compound have a low blood solubility lack of irritant action and marked analgesic action while inflamability and respiratory depresent action were its draw back (Conway 1965).

Although this agent had broad usefullness as an inhalation anaesthetic, in cardiac surgery, neuro-surgery, pediatric surgery and obstetric anaesthesia, but it became less popular due to almost simultanious introduction of Hallothane, which was synthesized in 1951 and examined by Suckling in 1951. It was a safer anaesthetic agent.

It became more popular due to the smooth and easy induction rapid recovery and absence of irritation and minimal nausea and vomitting and above all its noninflamability (Johnson 1956, Bryce Smith and Burns et al., 1957 and Abajain et al., 1959).

There was tremendous advancement in the field of anaesthesia with the discovery of muscle relaxant beside the inhalation agents.

Tubocurrarine was the first neuromuscular blocking agent which was first time discovered by King in 1935 but it was clinically used in 1942 by Griffith and Johnson (Griffith H.R. and Johnson G.E. 1942).

This drug was first used for abdominal surgery as a muscle relaxant in 1946 by Harroun in Britain (Harroun P. et al., 1946) and was established by T.C. Gray and John Halton of liverpool in great Britain (Gray T.C. 1946).

After the introduction and establishment of tubocurrarine the pharmacologist throughout the world sought for synthetic drugs with a similar action and properties to that of tubocurrarine.

As a result of this search gallamine triethiodide was synthesized and discribed by Ewvet in 1947 and used clinically by Huguennard and Bone in 1948, in France and by Mushin and his collegues in England in 1949 (Mushin W.W. et al., 1949).

After this there was the chain of the various muscle relaxant come into light day by day.

In 1948 thonium was described by Barlow and Ings and it was clinically used by Organe in 1949 (Organe G.S.W. et al., 1949).

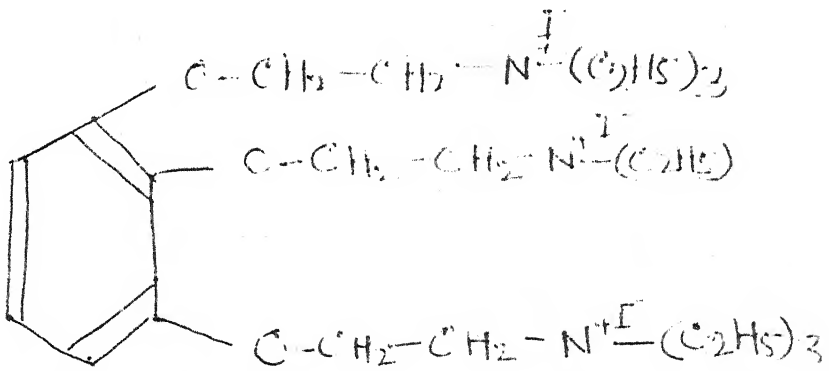
Danial Bovel and his coworker of Paris introduced two muscle relaxant one gallamine ethiodide in 1949 and other suxamethonium.

Suxamethonium was first used in anaesthesia by Von Daldel of Scottholm in 1951 and by Ottomayer Hoffer in Vienna and by Cyril Fredrich in Britain. Its effect in anaesthetized animal and man was described by Castillo and De Beer in 1950 and by Theself in 1951.

In 1947 Bovel and his coworkers described a synthatic muscle relaxant gallamine triethiodide. The effect of this relaxant in man was first described by Huguennard and Bone (1948) in France and by Mushin

and his colleagues (1949) in England.

Gallamine triethiodide is chemically tri (B-diethylaminoethoxy) benzine triethiodide. It is white amorphous powder, nonirritant relatively stable and available in 40mg/ml solution in 2ml and 10ml amples.



GALLAMINE TRIETHIODIDE

The intravenous bolus dose of gallamine triethiodide is most preferred route. The onset of action occur within 90-120 second and duration of action between 20-30 minutes. Supplementary doses of gallamine 20-40mg are given as required.

Gallamine triethiodide acts at the neuro-muscular junction by nondepolarization block. The currare molecule combine with the end plate receptors.

Gallamine distributed throughout the body and about 30-100% is excreted unchanged in urine within two hours (Mushin et al., 1949). Prolong paralysis may follow the use of gallamine in cases with poor renal function (Fairley 1950, Montgomery and Benett Jones 1956, Faldman and Levi 1963).

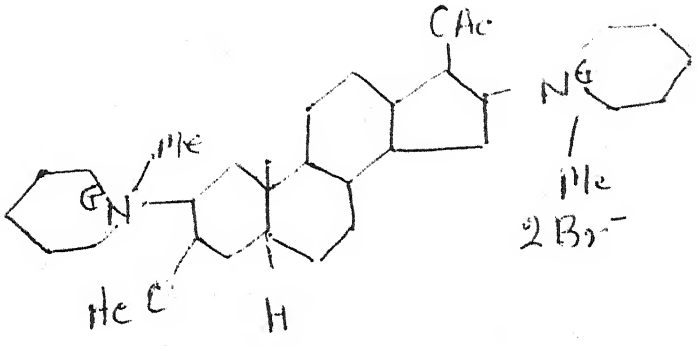
This is partly due to the loss of redistribution sites in the kidney and also due to the lack of alternative pathway of excretion for the drug (Faldman et al., 1969).

It is bound to serum albumine and the increase in potency by increasing the pH.

In 1956 W.D.M. Patton made distinction between the depolarizing and nondepolarizing muscle relaxant depending upon the mechanism of their neuromuscular blocking activity.

Pancuronium bromide was introduced in clinical anaesthesia by Baired and Reid in 1967. It is an odourless white crystaline powder, with a bitter test. It melts at 215°C with decomposition.

Chemically pancuronium bromide is a bisquart-ernary ammonium compound which is relatively stable and is supplied for clinical purpose in 2ml ample containing 2mg/ml.



PANCURONIUM BROMIDE

The intravenous bolus of pancuronium is the most prefered route onset of paralysis occur within 2 to 3 minutes. The paralysis produced by pancuronium last for about 25 to 45 minutes and a satisfactory 'topping up dose' is about 1/5 to 1/10 of the original paralysis dose.

Pancuronium acts at the neuromuscular function in man by nondepolarization (Baired and Reid).

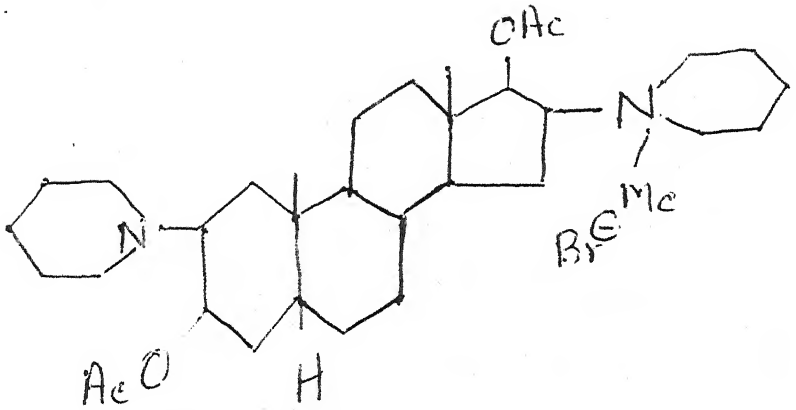
Pancuronium is believed to be excreted mainly unchanged in the urine but can be biodegraded to less active and inactive compound by metabolism up to 15% of injected dose of pancuronium may be recovered from urine as 3-hydrozy derivative. In the absence of renal excretion large amount of the drug can be recovered from the bile much in the form of steroid in which the 17 acetyle group has been hydrolysed to either the hydrogen or hydrozy derivative (Agoston, Kerston and Meifer 1973, Agoston et al., 1973, Somogyi Shanks and Triggs 1977).

Like all the muscle relaxants it is highly charged ion and is therefore unlikely to pass vital membrane easily. There is no evidence available in man that it is not believed to cross blood brain barrier. It has very little fat solubility.

There was no evidence suggested hystamine release after administration of pancuronium however alargic reaction have been reported (M.C. Dowel and Clark 1969, Nana et al., 1972).

Hugin and Kissling introduced Alcuronium in 1961 and Pancuronium was discovered clinically by Baired and Ried in 1967 but used in anaesthesia by Burkett W.R. et al., in 1968.

In 1979 Vecuronium was introduced by Durant et al., and was introduced in clinical use for the first time, by Crul and Booij of France in 1980 (Crul J.F. and Booij L.H.D. 1980).



CHEMICAL FORMULA OF VECURONIUM

Vecurronium bromide was originally known by its research code ORG NC 45 and was developed by David Savage of organon Technika, laboratories (Savage et al., 1980). Vecuronium was developed by nonhormonal properties of steroid molecule, which is an androstanyl derivative of acetylecholine.

Vecuronium bromide is a buffered freeze dried powder, available as 4mg per ample with 1ml ample of water for injection as solvent.

The powder can be kept for 3 years provided they are stored in the dark at a temperature below 25°C. The trade name of Vecuronium, norcuron reflects the fact that 'nor' indicates that Vecuronium has exactly the same chemical structure as Pancuronium except for the absence of a methyle group. The missing methyl group is one which is attached to the quaternary nitrogen atom which is itself attached to the 'A' ring of the steroid nucleus.

Vecuronium is a monoquaternary homologue of Pancuronium, having negligible ganglion blocking and vagolytic properties (Agoston et al., 1980).

The maintainance dose of Vecuronium is 0.03 to 0.05mg/kg body weight. The duration of effect is 10-20 minutes. However the larger dose of Vecuronium could lead to prolonged total duration of action. It is metabolized in the liver and mainly excreted in the bile, a small quantity is also excreted in the urine.

Neuromuscular effects of Vacuronium are potentiated by both respiratory and metabolic acidosis. The alkaline medium accelerate the decomposition of Vecuronium. The potentiating effect of hyperventilation on the duration of neuromuscular block are probably minimal in clinical practice.

Cumulative effects was not seen after repeated doses of Vecuronium. Vecuronium possesses an antimuscarinic action 1-3 times than Pancuronium.

As our country is a developing country so the ether is still in use along with several muscle relaxant due to the shortage of the sophisticated and costly instruments. As for as the safety of the patient is concerned with the use of these agents. Various experimental studies have been performed with a view to investigate the effect of anaesthesia on blood sugar and its mechanism because level of the blood sugar is the one

of the parameter for the safety of the patient.

This study perhaps, started with the experiment of Seeting in 1905 on the dog, in which there was rise in blood sugar level after introduction of diethyl ether and was confirmed by other workes.

That ether produces hyperglycaemia in animals is proved beyond doubt, but whether there is any significant effect in man is still debatable. Same is the state of other anaesthetic agents.

Before evaluating the changes in carbohydrate metabolism during anaesthesia, it will not be out of place to first consider some physiological aspect of carbohydrate metabolism.

PHYSIOLOGICAL ASPECTS OF BLOOD SUGAR

Glucose, the main conversion product of carbohydrate food, enters the blood from the intestine. Since it is a readily diffusible substance, is distributed fairly uniformly throughout the body fluid both extracellular and intracellular.

Glucose is metabolically inert and can not be utilized as such. Sugar is constantly added to blood and being utilized and stored. So maintainance of the blood sugar at a constant level is a balance between production and loss.

During the process of metabolism of glucose the source, storage and utilization of glucose come into play. Nervous influence, enzymatic action and harmonal involvement also have a significant role.

With a correct coordination between the different endocrine glands, several enzymes and hormones, blood sugar is maintained at constant level. The liver has an important role in this process.

Anatomically liver is the largest organ with numerous functions. Among the different functions during carbohydrate metabolism it plays two important roles-

1- By breaking down of glycogen it can liberate glucose into the circulating blood to maintain the blood sugar at its normal level.

2- It synthasies glycogen from glucose or other precursors and store it.

The glucose of the body arises from several sources-

1- The first is the intestinal absorption of the product of carbohydrate digestion.

2- Next from glycogenolysis from glycogen by an initial phosphorolysis which gives Glucose-1-phosphate and this product in the body rapidly comes into equilibrium with its isomer Glucose-6phosphate to give glucose and is governed by enzyme present in the liver.

3- Third source of glucose is from gluconeogenesis and the formation of glucose from noncarbohydrate precursurs. Among these are glucogenic aminoacids, glycerol and fatty acids arising from fats.

As in the process of glycogenolysis the liver produces the necessary enzyme to hydrolyse Glucose-6-phosphate to yeild glucose through gluconeogenesis.

Glucose absorbed from the gut is carried by the portal blood to the liver some of which is retained for conversion to glycogen. The reminder passes on to the systemic circulation for the use as follow.

1- It may be oxidised in the tissues.

2- It may be converted to fat.

3- It may be converted to glycogen in muscles, glycogen is formed in the liver. It consist of many hundreds glucose units linked together with the elimination of water. The formation of liver glycogen is sumerized in fig. 1

PROPERTIES OF LIVER GLYCOGEN

1. It is a suitable form to store carbohydrate.

2. Being insoluble it exerts no osmotic tension and so does not disturb the intracellular fluid content and does not diffuse from its storage site.

3. It has a higher energy level than a corresponding level of glucose.

4. It is the only readily avilable source for blood glucose.

5. It is radaily broken down under the influence of enzyme into glucose in the liver and maintains a constant blood sugar level to produce energy.

A high glycogen content in the liver depresses the rate of deamination and leaves the aminoacids for protein synthesis in the tissues. Similarly a higher level of glycogen prevents the breakdown of proteins and prevents ketosis.

The quickest way of building of liver glycogen is by raising the blood sugar level rapidly by I/V injection of glucose.

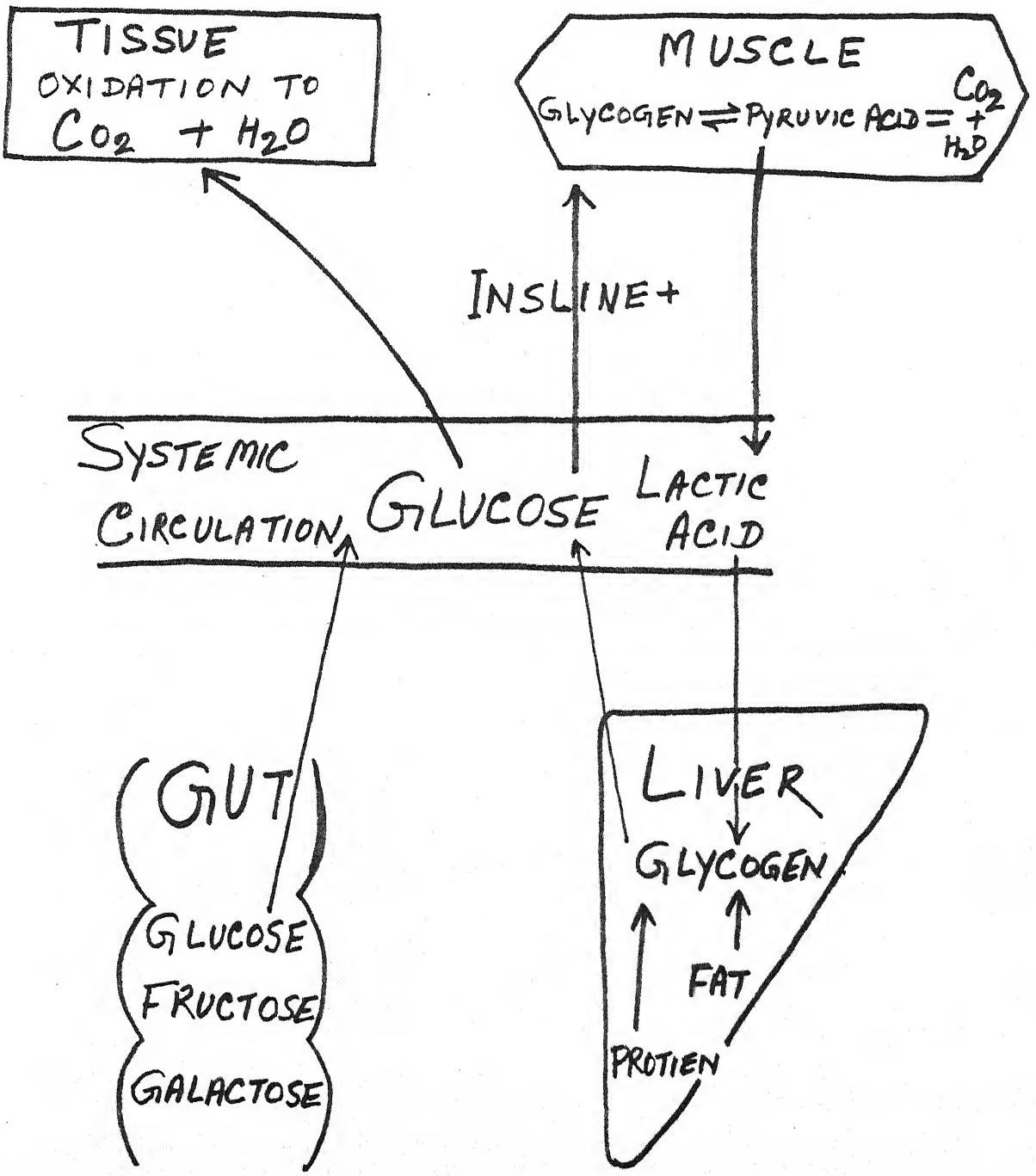
OXIDATION OF GLUCOSE AND GLYCOGEN IN THE TISSUES

In some tissues glucose from the blood is utilized directly for the provision of energy eg. Nervous tissue which obtain their energy by direct oxidation of glucose. It dependent upon the adequate level of circulating blood glucose because they have very low glycogen reserve. When the glucose is used up for energy it is first broken-down to pyruvate in the process of glycolysis and the pyruvate is then oxidised to Co_2 and water in the citric acid cycle (fig. 5).

CONVERSION TO FAT

Conversion of sugar to fatty acids involves the breakdown of carbohydrate to pyruvic acid and then

DIAGRAMATIC REPRESENTATION OF CARBOHYDRATE METABOLISM



FIG— 1

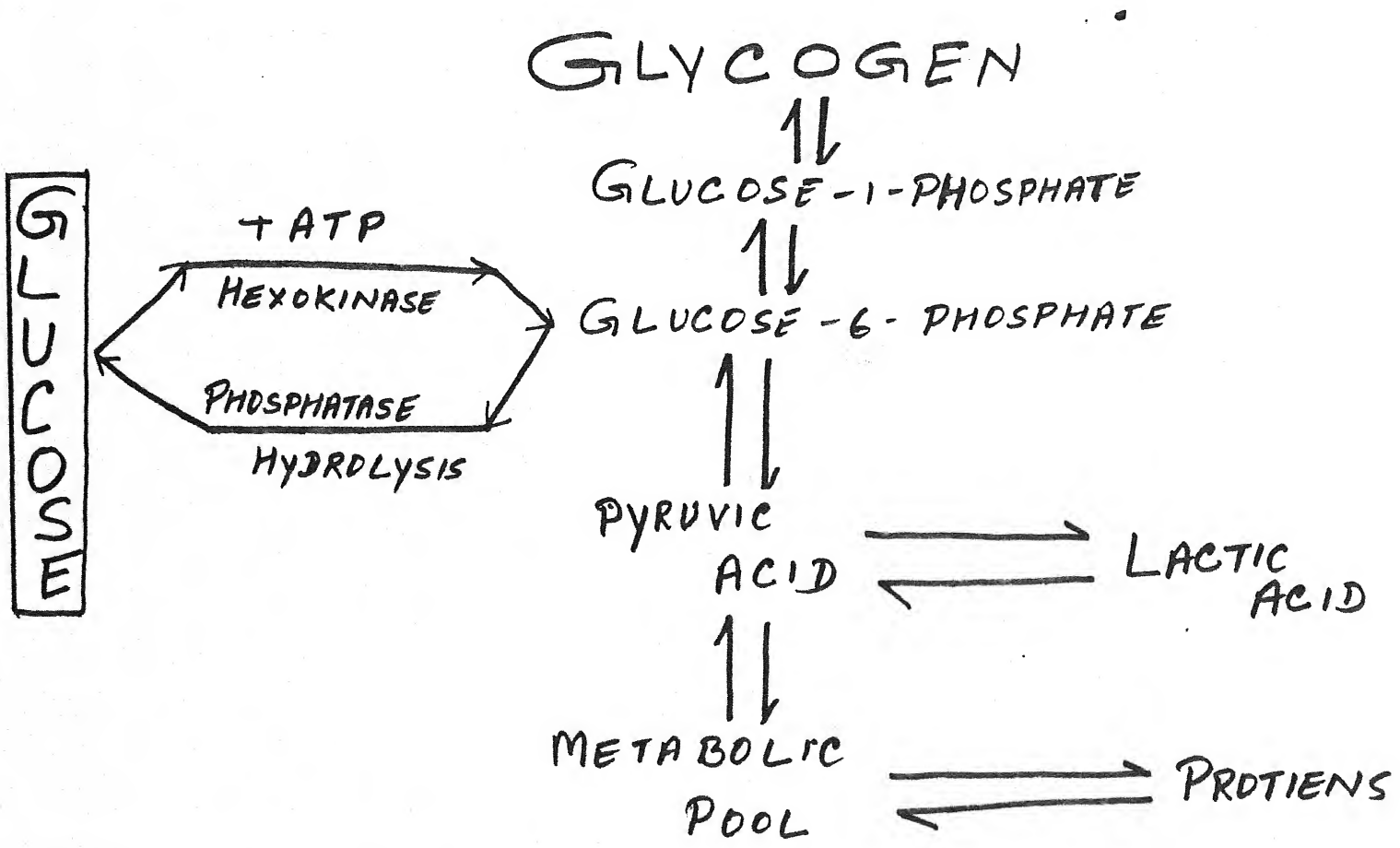


FIG. 2 SOURCES OF GLUCOSE

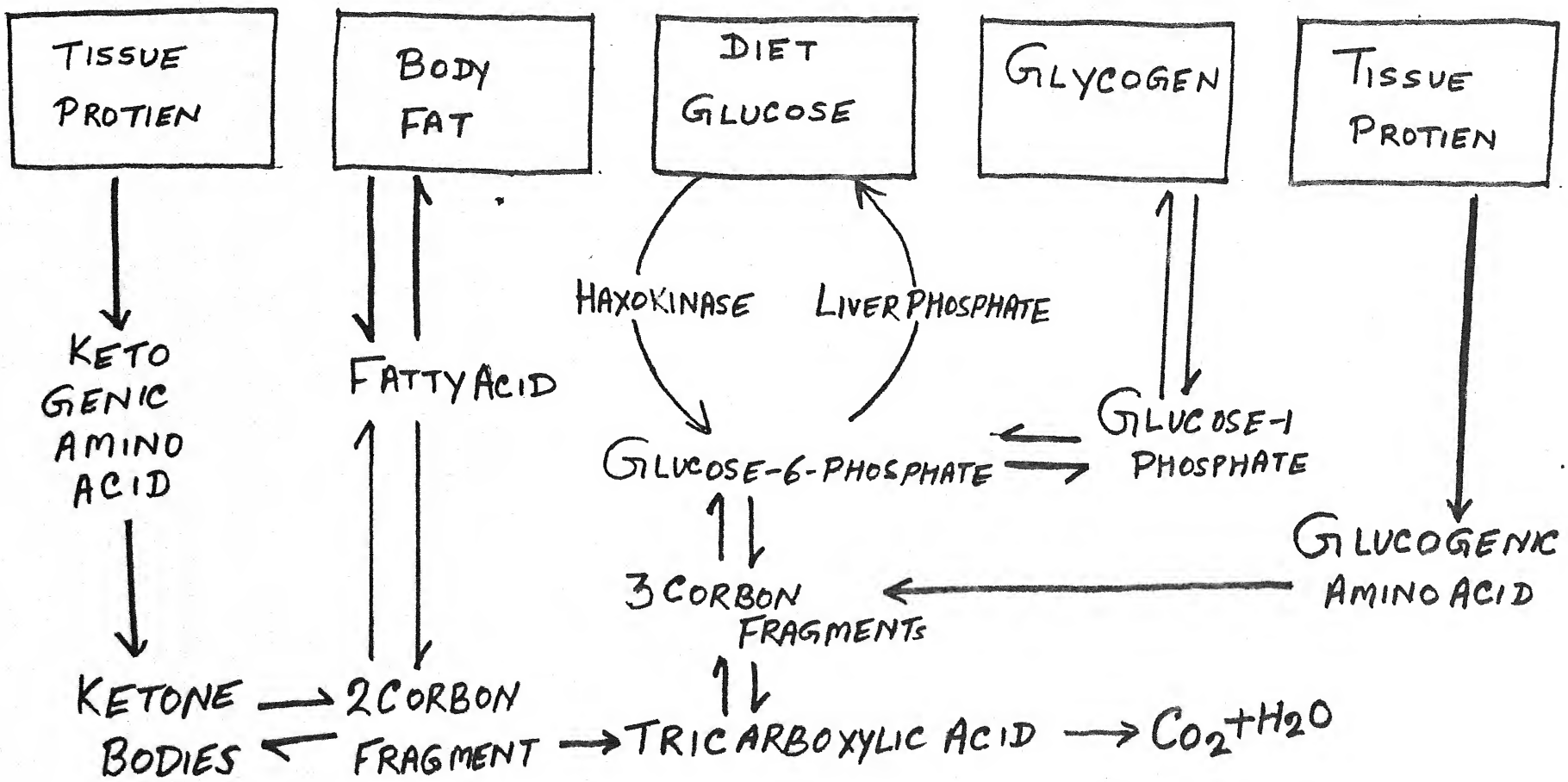


FIG 3 SOURCES of GLUCOSE.

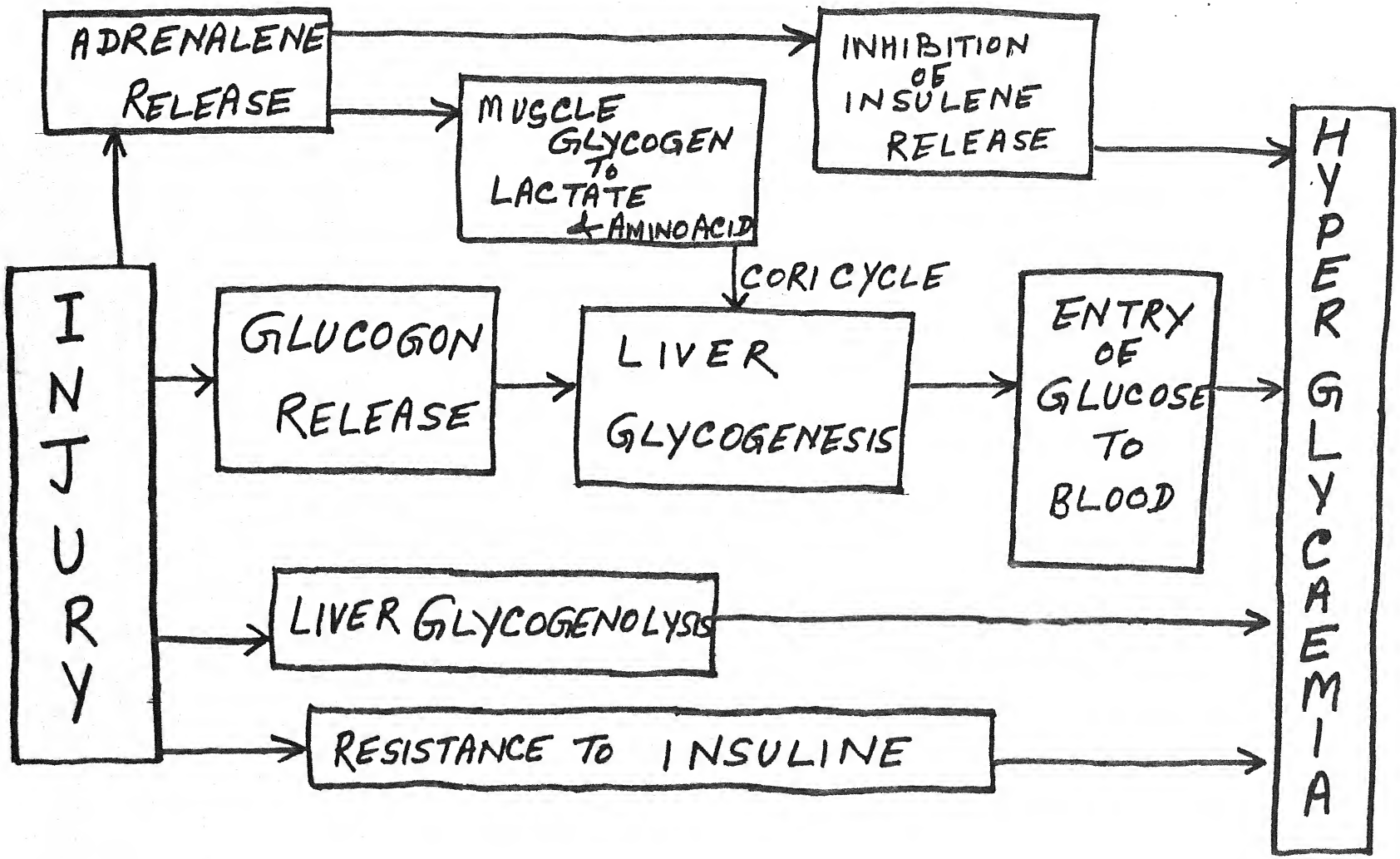

ADRENALENE RELEASE
INHIBITION OF INSULENE RELEASE
MUSCLE GLYCOGEN TO LACTATE & AMINO ACID
CORI CYCLE
INJURY
GLUCOGON RELEASE
LIVER GLYCOGENESIS
ENTRY OF GLUCOSE TO BLOOD
HYPER GLYCAEMIA
LIVER GLYCOGENOLYSIS
RESISTANCE TO INSULINE

FIG - 4

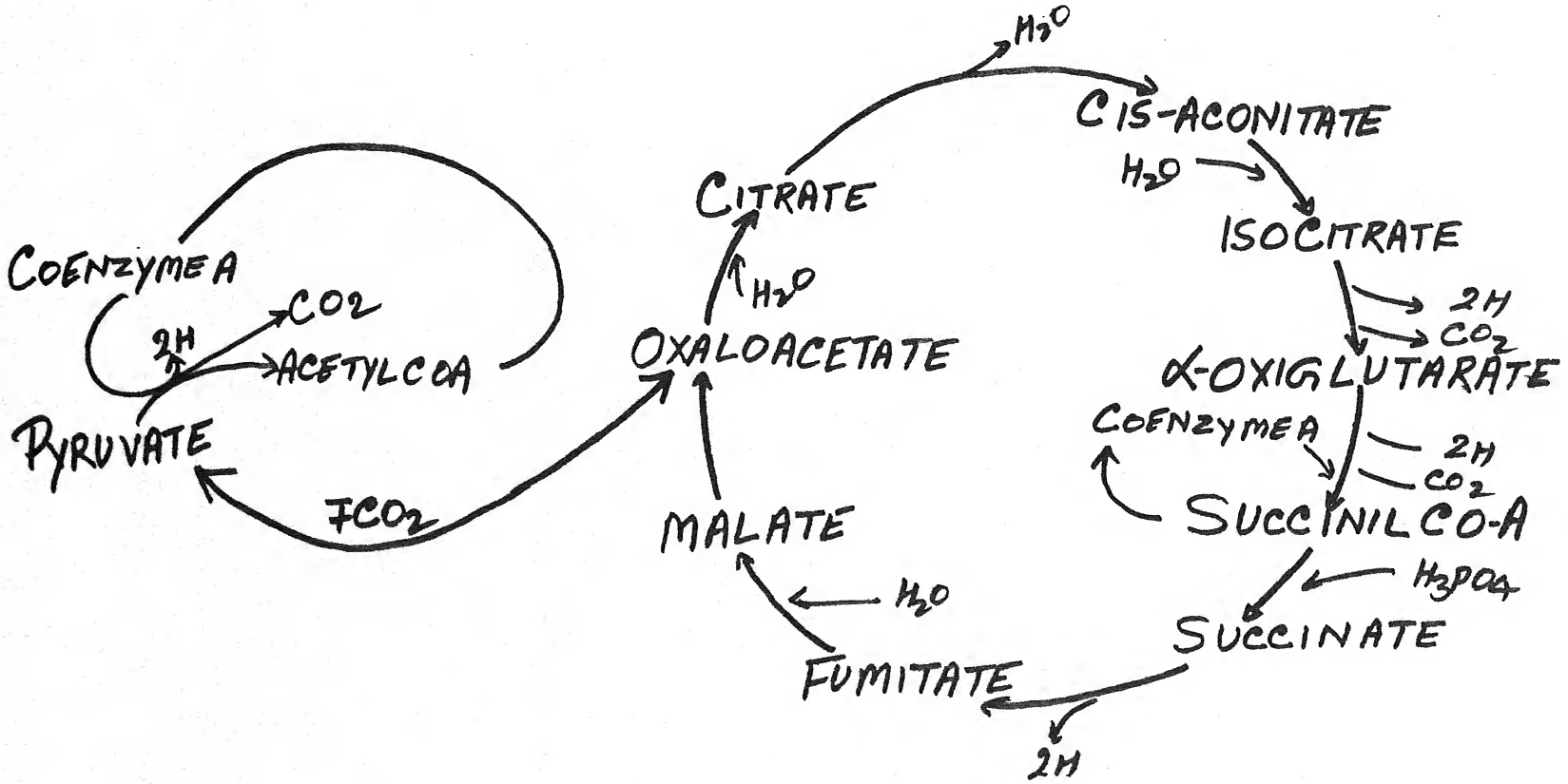

COENZYME A
2H
CO2
ACETYL CO A
PYRUVATE
CO2
OXALOACETATE
H2O
CITRATE
H2O
CIS-ACONITATE
H2O
ISOCITRATE
2H
CO2
α-OXIGLUTARATE
COENZYME A
2H
CO2
SUCCINIL CO-A
H3PO4
SUCCINATE
2H
FUMITATE
H2O
MALATE

FIG - 5

converted to acetyle CO-enzyme A. Molecules of CO-enzyme A are condensed together to long fatty acid chains which give rise to fat. This process is shown in fig. 3

MUSCLE GLYCOGEN

This is formed from the circulating blood glucose. The rate of formation of muscle glycogen is increased by a rise of blood glucose and by the presence of insulin. Muscle glycogen is consumed during exercise and is built up again from glucose at rest. Muscle glycogen can not be readily converted to blood glucose.

BLOOD GLUCOSE REGULATION

The normal morning fasting blood sugar level in an adult varies from 80-120mg per 100ml of blood. The concentration of glucose in human blood is the same in the cells as that in the plasma.

The blood sugar level is maintained at fairly constant level by a complex mechanism.

The main function of liver is building up and breakdown of glycogen rather than its storage, in carbohydrate metabolism. Under fasting condition when sugar is not reaching the blood from the alimentry canal the rate of glycogenolysis is equal to the rate

of blood sugar utilization and the blood sugar level is maintained relatively constant. At the same time gluconeogenosis start in the liver from noncarbohydrate material which make the hepatic glycogen store maintained when sugar is not available from G.I.T. The process of gluconeogenesis provides the liver glycogen from which the blood sugar is supplied.

In the muscle the blood sugar is built up into glycogen which gives rise to lactic acid and pyruvic acid during muscle contraction. Some of the lactic acid is diffused into the blood, reaches the liver and built up again into glycogen.

In absence of glucose supply from the gut the liver maintains the blood sugar. But when the supply from the gut is available the supply from the liver is temporarily cut off.

Insulin of pancreas controlls the out put or intake of glucose by the liver in response to variations in blood glucose. Adrenaline on the other hand, by slowing the rate of formation of muscle glycogen from of blood glucose and introducing resistance to the migration of blood glucose to tissue causes hyper glycemia.

Insulin is essential for the maintainance of blood sugar and life is incomplete without it.

Glucose is inert unless it undergoes phosphorytation to Glucose-6phosphate catabolized by the enzyme hexokinase. The insulin acts indirectly on hexokinase reaction.

In the anterior pituitary there is a specific inhibitor of hexokinase reaction when it is present in excess it slows the phosphorylation of Glucose-6-phosphate. Certain adrenocortical preperation prolong this inhibition.

An excess of insulin would overcome the physiologic inhibition of hexokinase reaction caused by the anterior pituitary inhibitor.

In presence of insulin, phosphorylation is accelerated and blood glucose level falls and all reactions of G-6-Po_4 would be accelerated eg. glycogenesis, lipogenesis etc.

Lack of supply of insulin or excess of anterior pituitary's inhibitor causes inpairment of hexokinase reaction. In reverse conditions the utilization of glucose is slowed down and this leads to rise in blood glucose level. The combustion of blood glucose soon

increases and glucose is no longer available for energy. Tissue protein have to be drawn which result in fall of liver glycogen causing ketosis and negative balance takes place.

Adrenaline and noradrenaline raise the blood sugar level by stimulating glycogenolysis. This rise in blood sugar level ensures a supply of carbohydrate from the muscular activity of fight or flight which follows an initial stimulus.

Adrenaline stimulate glycogenolysis in both liver and muscle causing hyperglycemia there by, a fall in liver glycogen. Administration of hydrocartisone cause a rise in blood sugar by stimulation of glycogenesis and to a diminished utilization of glucose.

Thyroid hormone has no role on blood sugar level.

There are many ways by which the nervous system brings about a rise in blood sugar:-

1. Stimulation through the hepatic nerves leading to the breakdown of glycogen already present in the liver.

2. Stimulation of internal secretion of adrenal gland.

3. Inhibition of internal secretion of insulin.

A much smaller quantity of adrenaline is required to raise the blood sugar.

When the requirements are more or less rate of production is regulated by alteration in the balance between the two antagonistic harmones, insulin and adrenatine in the blood. Adjustment in the balance between insulin and adrenaline is controlled by nerves.

As far as the effect of anaesthesia on blood sugar level is concerned various experimental studies have been performed. Seeling (1905) was perhaps the first person who has shown that the administration of ether in dog produces both hyperglycaemia and glycosuria by hampering with the action of insulin. However other anaesthetic agents did not interfare with insulin hyperglycaemia (Chamber's et al., 1927) or enhanced the effect of insulin (Aubertin and Tringuir 1932).

Thus the concept that anaesthetic might cause hyperglycemia by interfasting with the action of insulin in highly disputable.

Moreover ether did not produce its costomary rise of blood sugar in patient with hepatic disease (Cantarow and Gerhert 1931). This finding together points to the role of liver in the hyperglycaemia of ether anaesthesia.

It is more probable that the rise in blood sugar is the result of increased hepatic glycogenolysis either due to direct action of ether or due to increased H-ion concentration associated with ether anaesthesia.

Some observers have recorded considerable increase in the blood sugar level sometimes more than 200mg% (Mehler 1927 Mackary 1928, Mackay and Dyke 1928 Minitts and Huspers(1933) but Benerjee (1933) and Johnson (1949) have shown that this hyperglycaemia is the result of sympatheticoadrenal system with the subsequent release of epinephrine and mobilization of glycogen from the liver.

The demonstration of glucose intolerence during thiopentone anaesthesia appears to be relatively straight forward sympathoadrenal response. The blood sugar remains essentialy unchanged in the fasting patient anaesthetized by thiopentone alone (Olmsted and Coulhard 1928 and Ravdin 1929).

Cantarrow and Gehert (1931) have studied the effect of ether anaesthesia on patients having liver and biliary diseases and found that glycogenic function of liver is impaired and the ether did not produce its costummary rise of blood sugar in such patients because it is fairly well established that anaesthetics diminished the uptake of glucose by the tissue (MacIntosh and Pratt 1938).

Knofel (1936) advauces the theory that anaesthetic agents acting on the cerebral cortex causes hyperglycaemia by stimulating the sympathetic system and production of epinephrine as there is no rise in blood sugar level in cases of patients haveing bilateral adrenolectomy (Macraie 1931).

Thus the mechanism of producing hyperglycaemia appears to be due to a central stimulation which is transmitted to the adrenal gland through the sympathetic system and these glands by hormonal action cause glycogenolysis in liver. Later on Johnson (1949) also observed the same finding. The acceptable hypothesis which gain the strength that the anaesthetic agent bring about the release of adrenaline into blood stream from the suprarenal gland inturn accelerate the disintegration of liver glycogen and so mobilize glucose.

According to Engstrand and Friedberg (1945) parenchymatous organ especially the liver are injured under nacrosis during anaesthesia and this nacrosis causes the highest degree of hyperglycaemia due to overflow of the epinephrine from the adrenal gland.

MacIntosh and Pratt suggested that ether and other anaesthetic agents accelerate the breakdown of glycogen in the brain, liver muscle and other tissue of the body causing the increase the blood sugar level. According to these worker this is the compansatory mechanism means to minimize the effect of anoxia upon nervous tissue caused by anaesthetic.

Several procedure eg. high spinal anaesthesia above 4th thorasic segment (in which splenchnic nerve was blocked), denervation of adrenal, total sympathictonary use adrenergic blocking agents have been shown to prevent hyperglycaemia due to ether nacrosis (Phillip and Freeman 1933, Johnson 1949).

Bunker (1958) stated that the most widly known clinical metabolic disturbance is the rise of blood sugar level during anaesthesia. While Murdoch (1958) said that anoxia itself causes most rapid rise in blood sugar level. Of the several hypothesis which is accepted

by many, is the hyperglycaemia occuring due to glycogen olysis in the liver as a result of liberation of catcholamine, perticularly adrenaline, mainly from the suprarenal gland and also from the adrenergic nerve endings, due to the central activation of sympathetic system, caused by stress response during anaesthesia and surgery (Keating 1958, Annannumthodo Keating and Patrick 1958).

Haris (1959) has shown that with unpremedicated patients the induction of anaesthesia may cause considerable emotion and stress and consequently cause rise in blood sugar level. With this view Hunter 1959) carriedout the experiment on blood sugar level and he concluds that during Halothane anaesthesia which is known to depress the sympathatico adrenal system there was no significant rise in blood sugar level even after 2 hours (Topkins and Artusio 1959).

Brewster, Bunker and Beecher (1962) concluded that total pre ganglionic sympathatic block prevented epinephrine and nor epinephrine output during ether anaesthesia and the absence of metabolic acidosis and hyperglycaemia was due to the failure of catecholamine release.

The well known elevation of serum lactate and pyruvate and the less consistant elevation in citrate and alpha ketoglutarate which occur during ether anaesthesia but not with thiopentone may again be reasonably assumed to be related to increase sympathoadrenatine activity.

Irrespective of anaesthesia higher rise of blood sugar occured in those patients who under went abdominal and pelvic operation than in those undegoing extra abdominal operation.

This might be due to handling of viscera which is known to cause sympathoadrenal stimulation (Griffith 1953).

Annamunthodo et al (1958) attributed the rise in blood sugar level to the mobilization of glucose from liver while Griffith (1939) has observed that rise in blood sugar level was propotioned to the blood level of advenaline.

Clark (1968) showed rise in blood sugar during body surgace surgery with thiopentone nitrous oxide with or with/out relaxant. He observed that general anaesthesia itself did not show any elevation of blood sugar till the commencement of surgery and though that stress of

surgery increases circulating catecholamine and corticosteroid which inturn increase the blood sugar level.

Mehta and Buston (1975) postulated that surgical stress raises blood glucose which inturn is due to the activation of the hypothalamopituitary-adrenal axis and anaesthesia definitly modifies the effect of surgical stress.

The attenuation of blood glucose level changes varies with the anaesthetic technique (Amanunthodo et al., 1958, Browage et al., 1971 and Aheram and Walker 1979) depending upon their ability to supress the reflex sympathatic activity in response to surgical stimulation.

Any form of stress is accompanied by change in the level of plasma cortisol catecholamine growth hormone, insulin and glucagon (Oyarma and Katsuki 1970).

The stress of surgical intervention has 3 component:-

1- The psychic stress due to fear of impending operation.

2- Stress due to anaesthesia.

3- Stess due to surgical trauma.

Preoperative psychic stress is reported to cause a significant elevation of blood sugar level regardless preoperative starvation (Allison et al., 1969).

R. Sudhina Laxmi, M. Usha Rani, D. Vijays Kumar Menon and M. Venketa Rao (1973) also observed in their work that there was a rise in blood sugar level after induction and during surgery. Ether showed hyperglycaemic tendency and maximum rise in blood sugar was observed in patients operated under ether anaesthesia.

As far as the effect of muscle relaxant is concerned its effect on blood sugar level during anaesthesia not much work have been done but it is clear that the muscle relaxant have no direct effect on the blood sugar physiology. Any change in the blood sugar level may be due to the other factors like anxiety, hypoventilation or hyperventilation during anaesthesia and lighter plain of anaesthesia.

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OBSERVATION

OBSERVATION

In the present study the effect of ether, pancuronium, gallamine and vecuronium on blood sugar level was compared and evaluated in 60 patients.

Patients were of both sexes and over the age of 20 years but below the age of 60 years.

These 60 patients were randamly alocated into four groups depending upon the anaesthetics or muscle relaxant used. Each group was comprised of 15 patients.

AGE DISTRIBUTION

Out of 15 patients in group I (Ether) 4 patients were between age group of 20-30 years, 5 patients between 30-40 years, 3 patients between 40-50 years and 3 patients between the age group 50-60 years.

In group II (Pancuronium) 5 patients in range of 20-30 years 2 patients in range of 30-40 years of age. 3 patients were in age group of 40-50 years and 5 patients between 50-60 year of age group.

TABLE NO. 1

SHOWING THE AGE DISTRIBUTION OF THE PATIENTS

Age group	I(E) %	II(P) %	III(G) %	IV(V) %	Total
20-30	4	5	3	3	15
30-40	5	2	1	7	15
40-50	3	3	5	2	13
50-60	3	5	6	3	17
Total	15	15	15	15	60

In group III (Gallamine out of 15 patients 3 patients were between the age group of 20-30 years 1 patient was between 30-40 year 5 patients were between 40-50 years of age group while 6 patients were in age group of 50-60 years.

In group IV (Vecuronium) out of 15 patients 3 patients were in between 20-30 years of age 7 patients in age of 30-40 years 2 patients were between 40-60 year of age while 3 patients were in the age group of 50-60 years.

Regardless the anaesthesia group there were 15 patient in age group of 20-30 years and 30-40 years each. 13 patient were in age group 40-50 years. While 17 patients in the age group of 50-60 years.

SEX DISTRIBUTION

Out of 60 patients 33 patients (55%) were male patients while remaining 27 patients (45%) were female patient.

TABLE NO. 2

SHOWING SEX DISTRIBUTION

Sex	Ether	Pancuronium	Gallamine	Vecuronium	Total
Male	8	4	9	12	33(55%)
Female	7	11	6	3	27(45%)
Total	15	15	15	15	60

Distribution of male and female patients in all 4 groups was as follow:-

8 male and 7 female patients was in ether group while 4 male and 11 female were in pancuronium group 9 male and 6 female were in gallamine while remaining 12 male and 3 female patients were in vecuronium group (Table no.2).

Table no. 3 shows the type of operation in which the drug were used. In Ist group under ether anaesthesia cystolithotomy were performed in 3 patients hysterectomy in one patient tonsilectomy in 2 patients

laperatomy in 3 patients cholecystactomy in one patient mastoidectomy in 2 patients nephrolithotomy in one patient and mastactomy in 2 patients.

In second group pancuronium was used to fascilitate for hysterectomy in 3 patients laperectomy in 5 patient cholecystectomy in 4 patients and mastoidectomy in 3 patients.

TABLE NO. 3

SHOWING THE VARIOUS TYPE OF SURGICAL PROCEDURE

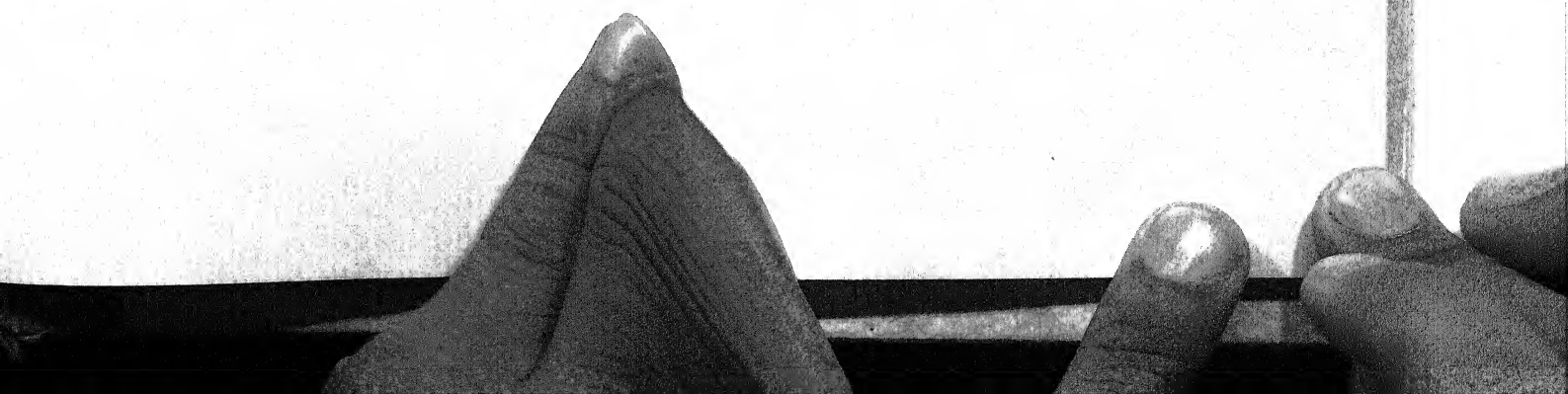
S.No.	Name of operation	I(E)	II(P)	III(G)	IV(V)	Total	%
1	Cystolithotomy	3	-	-	1	4	6.66
2	Hystrectomy	1	3	1	2	7	11.66
3	Tonsilectomy	2	-	2	1	5	8.33
4	Laperatomy	3	5	4	5	17	28.66
5	Cholecystactomy	1	4	1	3	9	15.00
6	Mastoidectomy	2	3	2	1	8	13.33
7	K.Nailing	-	-	3	1	4	6.66
8	Nephrolithotomy	1	-	1	1	3	5.00
9	Mastactomy	2	-	1	-	3	5.00
	Total	15	15	15	15	60	100.00

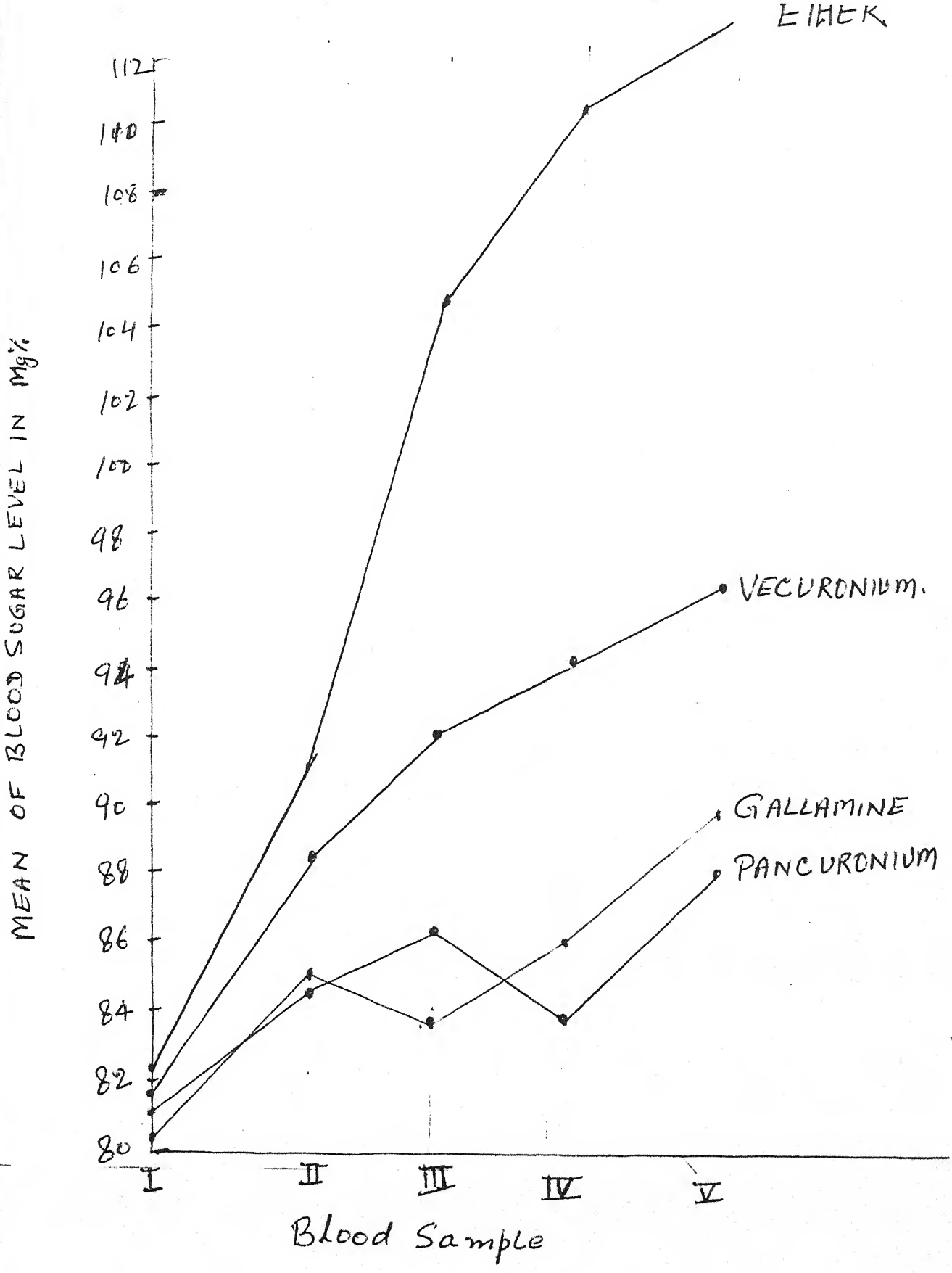
In group III muscle relaxation was provided with gallamine for hysterectomy in one patient tonsilectomy in 2 patient laperatomy in 4 patients cholecystactomy in one patient mastoidectomy in 2 patients K-Nailing in

3 patients Nephrolithotomy in one patient and mastactomy in one patient (Table no. 3).

In IVth group relaxation was given with the help of vecuronium for cystolithotomy in one patient hysterectomy in 2 patients tonsilectomy in one patient laperatomy in 5 patients cholecystectomy in 3 patients, mastoidectomy, K-Nailing and nephrolithotomy, 1 patient each (Table no.3).

In this way there were 4 patient of cystolithotomy (6.66%) 7 patients of hysterectomy (11.66%) 5 patients of tonsilectomy (8.33%) 17 patients of laperatomy (28.66%) 9 patients of cholecystactomy (15%) 8 patients of mastoidectomy (13.33%) 4 patients of K-Nailing (6.66%) 3 patients of nephrolithotomy (5%) and 3 patients of mastactomy (5%)(Table no.3).




ETHER
VECURONIUM.
GALLAMINE
PANCURONIUM
112
110
108
106
104
102
100
98
96
94
92
90
88
86
84
82
80
I
II
III
IV
V
MEAN OF BLOOD SUGAR LEVEL IN Mg%
Blood Sample

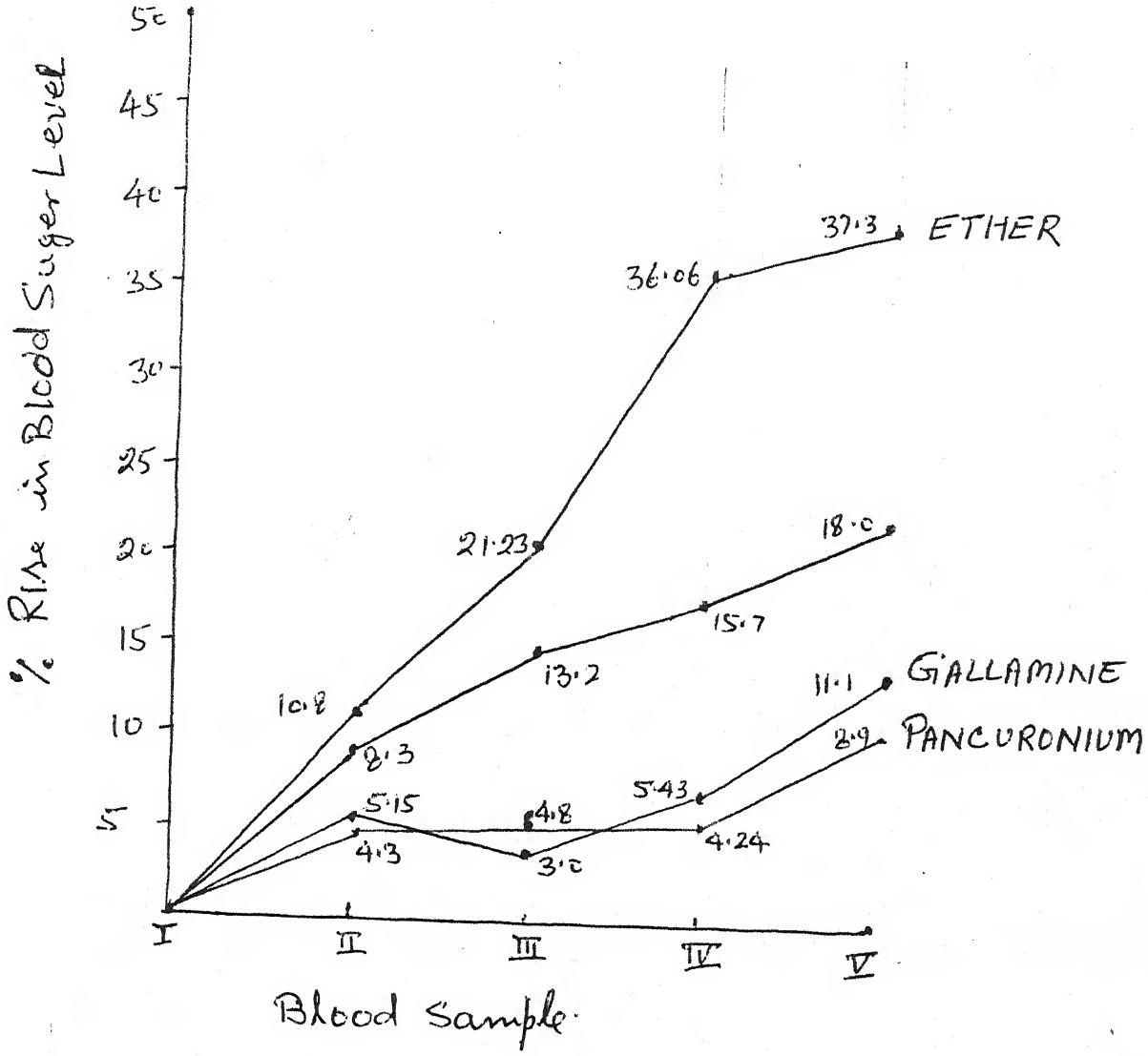

% Rise in Blood Suger Level
50
45
40
35
30
25
20
15
10
5
I
II
III
IV
V
Blood Sample
ETHER
GALLAMINE
PANCURONIUM
10.2
21.23
36.06
37.3
8.3
13.2
15.7
18.0
5.15
4.8
4.3
3.0
5.43
4.24
11.1
8.9

TABLE NO.6

SHOWING THE DIFFERENT STATISTICAL DATA(VALUES) FOR PANCURONIUM GROUP

	I	II	III	IV	V
Mean	81.2	84.7	85.06	83.2	88.46
S.D.	28.86	42.01	59.38	44.78	35.26
Mean% gain		4.32%	4.82%	4.24%	8.96%
$\bar{Z}$		2.2	4.6	5.6	6.26
σ_z		3.5956	4.80	3.77	5.7258
t value		2.37	3.711	5.705	4.04
P value		<0.03	<0.027	<0.01	<0.01

TABLE NO.7

SHOWING THE DIFFERENT STATISTICAL DATA(VALUE) FOR GALLAMINE GROUP

	I	II	III	IV	V
Mean	80.73	84.9	83.13	85.13	95.73
S.D.	25.231	23.50	37.41	24.64	32.37
Mean% gain		5.16%	3.01%	5.43%	11.1%
$\bar{Z}$		2.9	2.9	6.199	5.06
t value		4.673	4.233	6.1538	9.2836
P value		<0.01	<0.01	<0.025	<0.05

TABLE NO. 4

SHOWING THE VALUES IN TERMS OF MEAN ± STANDARD DAVIATION

Sample	Ether	Pancuronium	Gallamine	Vecuronium
I	82.5 ±38.20	81.2 ±28.86	81.73 ±25.23	81.66 ±33.33
II	91.4 ±27.118	84.7 ±42.01	84.9 ±23.50	88.46 ±22.58
III	105.86 ±26.09	85.06 ±59.36	83.13 ±37.41	92.13 ±24.35
IV	111.93 ±19.63	83.02 ±44.74	85.13 ±24.64	94.53 ±24.38
V	114.33 ±28.4178	88.46 ±35.26	89.73 ±32.37	96.26 ±22.85

TABLE NO.5

SHOWING THE DIFFERENT STATISTICAL DATA(VALUES) FOR ETHER GROUP

	I	II	III	IV	V
Mean	82.5	91.4	105.86	111.93	114.33
S.D.	38.20	27.118	26.09	19.63	28.4178
Mean% gain		9.45%	20.36%	26.30%	28.48%
$\bar{Z}$		6.93	23.6	28.86	30.6
δz		6.06	11.23	10.09	9.0079
t value		4.406	8.1328	11.069	13.14339
P value		<0.001	<0.001	<0.001	<0.001

TABLE NO.8

SHOWING DIFFERENT STATISTICAL VALUES FOR VECURONIUM GROUP

	I	II	III	IV	V
Mean	81.66	88.46	92.46	94.53	96.26
S.D.	33.33	22.58	24.35	24.36	22.85
Mean%gain		8.31%	13.224%	15.751%	18.0%
$\bar{Z}$		3.866	7.666	9.93	11.666
$\not{\delta}z$		4.97	4.66	4.57	4.391
t value		2.356	5.1618	6.7329	7.932
P value		$\not<$0.03	$\not<$0.01	$\not<$0.025	$\not<$0.01

ANALYSIS OF DATA

The results obtained from the all four groups were compared by using simple statistical method. The t test was used to compare between each group and 'P' value was taken from chart of probability.

STATISTICAL CALCULATION

(1) Mean $\bar{X} = \frac{\Sigma X}{n}$

where X = values in mg%

n = No. of patients in the group

(2) Standard deviation (S.D.)

$$S.D. = \frac{(\Sigma X - \bar{X})^2}{n}$$

were X = value in mg%

$\bar{X}$ = Mean

n = No. of patients

$$(3) \quad t \text{ value} = \frac{\bar{Z} \sqrt{n}}{\bar{Sz}}$$

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DISCUSSION

DISCUSSION

The present study was performed to evaluate the change in blood glucose level due to the effect of ether, and 3 muscle relaxant Pancuronium, Gallamine and Vecuronium when they are used for general anaesthesia. In the present study only adult patient were selected. Their age ranged between 20-60 years to circumvent the variables at the extrems of age. Patients subjected to routine surgical procedures were included in this study and emergency procedure were excluded to maintain standard condition as for as possible. All patients were of ASA grade I or II.

In this study same premedication drugs, induction agents and supplementary analgesia drugs were used in all cases to avoid the influence on the dosage and action of Ether or any muscle relaxant drug.

The present study shows that in ether group, when we take the mean of the value of blood sugar level of all 15 patients then we see that there was 10.8% rise in II sample when it is compared with Ist sample (control sample). While in III sample rise was 21.23% in IVth sample rise was 36-06% and in the Vth sample

this rise was 37.3% of the control (sample) value (Table No. 5).

In case of Pancuronium group this rise in IInd sample was 4.32% of the Ist (control) sample. In the same way when compared with control sample this rise was 4.82%, 4.24% and 8.96% in sample III, IV and Vth respectively (Table No. 6).

In case of Gallamine the rise in blood glucose level in terms of percentage of control value was 5.15% in IInd sample 3.001% in III sample 5.43% in IVth sample and 11.1% in Vth sample (Table No.7).

In case of Vecuronium the rise in blood glucose level in terms of percentage of control value was 8.3% in II sample 13.2% in III sample 15.7% in IV sample and 18% in Vth sample.

As shown in table no.5-8 we can compare the rise at different interval during the use of different drugs.

After premedication rise in blood sugar level is below 10% in all case wise ether, pancuronium, gallamine and vecuronium. But after 5 minutes of intubation there was above 20% rise in case of ether while in case of pancuronium and gallamine it was

below 5% and in case of vecuronium where it was above 13% rise when compared with control value.

After 30 minutes of intubation the percentage rise in case of ether was above 35% while in case of pancuronium and gallamine it was around 5% except in case of vecuronium where it was about 15%.

After extubation the percentage rise in case of ether was above 37% while in case of 3 relaxants it was about 9%, 11% and 18% respectively.

The rise in blood sugar level upto the pre-medication is almost equal in all four group of this study.

It is well known fact that preanaesthetic medication alone does not completly prevent the effect of preoperative psychic stress on blood glucose.

Any form of stress is accompained by change in the level of cortisol catecholamine growth hormone insuline and glucogone (Oyama an Matsuki 1970) which are intimatly associated with the regulation of blood glucose.

Increase in blood glucose concentration has long been known to occur after surgery trauma or anaesthesia as a result of sympathoadrenal stimulation (Mehta and Burton 1975). The stress has 3 component

(1) the psychic stress due to fear of impending of operation.
(2) stress due to anaesthesia.
(3) stress due to surgical trauma (Engquist and Wither 1972).

In this way the stress factor which is common in all four groups of this study could not be elementated completely even with the preanaesthetic medication. In this way the rise in blood glucose level in all groups, more or less is a must where this stress is responsible for such elevation of blood glucose level.

Although this rise varies with different groups for example there is 10.8% rise in case of ether group 4.32% in pancuronium group 5.15% in gallamine group and 8.3% in case of vecuronium group. But after start of surgery and use of different anaesthetic drug like ether pancuronium, gallamine and vecuronium there is steep rise in blood glucose level upto about 40% of value which was taken after premedication in case of ether group.

While it is between 5-16% in other three group. Other factors like surgical trauma and stress being common. There is only high rise in case of ether group and not so much in other relaxants group. This gradual increase in the blood glucose level in case of ether from 10.8% to 37.3% may be due to the use of ether because in other relaxant groups, this rise is below 18% which may be due to the surgical trauma and stress.

But in case of ether there is marked and prograssive rise in blood sugar level because the mean blood sugar level at the time of induction was 91.4mg% which increase upto 114.33mg%. There was change of 23mg% in blood glucose level. Ostama et al 1971 reported a prograssive rise in blood sugar level due to ether anaesthesia.

Similar findings was observed in this study during ether anaesthesia and surgery.

In case of pancuronium group there is little rise in blood glucose level before the use of relaxant i.e. about 4.32% rise, but after the use of relaxant there is very little change in blood glucose level that was upto only about 8.96-4.32 = 4.64%.

In this group total mean of blood glucose level was 84.2mg% which was at the time of induction and this increases upto only 88.46mg% at the end of surgery and anaesthesia. Thus the only change in blood glucose level was 88.46-84.20 = 4.26mg% (Table No. 6).

In case of gallamine at the time of induction the rise in blood glucose level was 5.15% which rises upto 11.1% at the end of surgery and anaesthesia.

There was mean blood glucose level 84.90mg% at the time of induction which rises upto 89.73mg% at the end of surgery and anaesthesia. It means there is only change of 4.83% during the whole process of anaesthesia surgery. In the similar way in case of vecuronium at the time of induction the mean blood sugar level was 88.66mg% which reaches upto 96.26mg% causing the only differance of 92.26-88.66 = 7.60mg%.

As for as no work regarding the effect of muscle relaxant on blood glucose level is available so in absence of the findings of previous work this is not possible to compare with the findings of present study.

In this way the change in blood glucose level due to the influence of ether and other 3 muscle relaxant was as under:-

Group I Ether - 23.00mg% or 40%

Group II Pancuronium - 4.26mg% or 4.64%

Group III Gallamine - 4.83mg% or about 6%

Group IV Vecuronium - 7.80% or 5%

This shows that there is very highly significant rise in blood glucose level during the ether anaesthesia while in case of relaxants there is only significant rise in blood sugar level.

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SUMMARY AND CONCLUSION

SUMMARY

The present study entitled "The study of changes in blood glucose level during general anaesthesia,in patients under going surgery" was carried out at M.L.B. Medical College Hospital Jhansi over a period of one year. The patients were divided into four groups depending upon the use of anaesthetic drugs.

For anaesthetic drugs were used:

1. Ether — Inhalational agent
2. Pancuronium
3. Gallamine — Non depolarising agents
4. Vecuronium

Ether is in use for a long period as a inhalational agent in the form of ether vapour. In the developed countries it is not in use now a days but in India being a devloping country it is still in use.

Non depolarising muscle relaxants revolutionized the anaesthetic practice. These drugs produce active muscular relaxation without unwanted side effects. They have made it possible for the anaesthesiologist to have adequate control over ventilation of the patients.

There is a clinical need for a new non-depolarizing muscle relaxant of shorter duration less cumulative property and fewer side effects. These new muscle relaxant will no doubt changes our pattern of practice by improving the safety and clinical relaxation.

ETHER

It was prepared by Valerius Cordus in 1540 and Sigmund August Forbenisius named it ether and was used in anaesthesia by W.E. Clarke in 1842.

It is volatile liquid of molecular weight 74 and boiling point 35°C specific gravity of vapour being 2.6. Ether vapour is flammable in air. Chemically it is inert and can be stored in dark cool place unaltered in body and 85-90% is eleminated by lungs. This causes increases in heart rate at first due to (i) Catecholamine libration (ii) Sympathatic stimulation and Vagal depression later the heart rate is relatively unchanged.

There are some side effect of ether.

1. Nausea and vomiting post operatively.
2. Explosive risk when used with O_2 and N_2O.
3. Slow induction and recovery from anaesthesia.

PANCURONIUM BROMIDE

Pancuronium bromide is a bisquaternery aminosteroid, described by Buckett, Hewett and Savage in 1967. It appeared to be an effective long acting non-depolarising neuromuscular blocking agent, without the evidence of steroid activity. The paralysis remained between 25 to 45 minuts.

Pancuronium is a markedly cumulative drug. It causes very little hystamine releare and mild transient depression of plasma cholenesterae. Pancuronium is known to have no effect on the liver or any influence over the carbohydrate metabolism causing no significant change in blood sugar level.

GALLAMINE TRIETHIODIDE

Gallamine triethiodide a synthetic nondepolarising muscle relaxant was first described by Bovet et al in 1947 and used in anaesthetic practice by Huguenard et al in 1947.

Chemically Gallamine is tri (B-diethyl amino ethoxy) Benzine tri ethiodide. It is intermediate acting agent with duration of action between 20-30 minutes.

Tachycardia and hypertension occur in the use of this drug but it has no direct or indirect effect over the liver function or carbohydrate metabolism.

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Vecuronium with its remarkable lack of effect on C.V.S. liver and other organ, is probably an example of the forerunner to a new generation of neuromuscular blocking agents which provide the anaesthetist with safer and more efficient means of producing muscle relaxation.

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The present study was planned with aim to study the changes in blood glucose level during general anaesthesia in patients under going surgery under ether, pancuronium bromide, gallamine triethiodide and vecuronium bromide.

A total number of 60 patients of either sex and between 20-60 years of age were selected to fulfil the purpose of study.

All the patients were of ASA grade I or II and they were randomly allocated in 4 groups on the basis of ether and 3 muscle relaxant used.

Which were as follow:-

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Induction was performed with the sleep dose of 2.5% thiopentone (4-6mg/kg) intraveinously followed by the administration of suxamethonium (80-100mg) I/V. Subsequently intubation was done with proper sized cuffed endotracheal tube connection were made to attach the patient with mapelson A circuit of Boyle's apparatus, I.P.P.V. was continued. Anaesthesia was maintained with the mixture of N_2O and O_2 (60:40) total gas flow was 7-9 litre/minutes.

OBSERVATION AND ANALYSIS

Patients in all the 4 groups were observed for the change in the blood sugar level at the different interval during anaesthesia. For this blood sample were collected as follow.

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from all the patients of each group. Blood glucose level estimation was done in each sample.

After the estimation of the blood glucose level in the various sample of the patients of all 4 groups and after analysing these findings with the help of statistical calculation, it was observed that there is very highly significant rise in case of the ether anaesthesia. This rise in upto 40% of the initial value.

But in case of pancuronium anaesthesia there is very small rise in the blood sugar level was observed. This case of gallamine a small rise was observed in blood sugar level which was 5 to 11%.

Lastly in case of vecuronium bromide anaesthesia there was upto 5% rise in blood glucose level.

CONCLUSION

From the present study we concluded that in comparisation of muscle relaxant ether causes much more hyperglycaemia during general anaesthesia which is very highly significant rise.

But in case of muscle relaxant the change in blood glucose level is just significant.

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ETHER

S.No.	Blood Glucose level in mg%					II-I	III-I	IV-I	V-I
	I	II	III	IV	V	A	B	C	D
1	96	100	108	108	120	4	12	15	24
2	76	80	112	122	122	4	48	46	46
3	116	118	135	138	140	2	19	22	24
4	103	112	128	132	136	9	25	29	33
5	100	106	128	130	132	6	28	30	32
6	76	76	87	94	97	6	11	18	21
7	92	96	115	126	128	4	23	34	36
8	104	110	122	136	136	6	18	32	32
9	103	112	138	140	140	9	35	37	37
10	42	48	61	71	67	6	19	29	25
11	72	90	104	105	108	8	32	33	36
12	55	86	90	92	95	13	35	37	40
13	36	40	48	52	54	4	12	16	18
14	110	136	140	152	150	25	30	42	40
15	65	63	72	78	90	-2	07	13	15
Total	1238	1371	1588	1679	1715	104	354	433	459
Mean	82.5	91.4	105.86	111.93	114.33				

PANCURONIUM

S.No.	Blood Glucose lavel in mg%					II-I	III-I	IV-I	V-I
	I	II	III	IV	V	A	B	C	D
1	104	98	122	114	118	-6	18	10	14
2	100	102	118	116	121	2	18	16	21
3	116	118	122	132	128	2	6	16	12
4	88	100	96	106	108	2	8	18	20
5	110	116	116	120	115	6	6	10	5
6	114	112	115	120	120	-2	1	6	6
7	56	60	62	66	80	4	6	10	24
8	72	76	74	80	82	4	2	8	10
9	122	128	130	133	135	6	8	11	13
10	66	62	76	81	80	4	10	15	14
11	76	80	83	88	88	4	7	12	12
12	52	56	58	55	60	4	6	3	8
13	42	48	46	53	54	6	4	11	12
14	33	35	39	42	42	2	6	9	9
15	108	111	116	112	112	3	8	4	4
Total	1261	1226	1156	1218	1327	33	114	159	184
Mean	84.06	81.7	77.06	81.2	88.46				

GALLAMINE

S.No.	Blood Glucose level in mg%					II-I	III-I	IV-I	V-I
	I	II	III	IV	V	A	B	C	D
1	76	76	84	117	120	0	8	21	24
2	107	112	110	122	127	5	3	15	20
3	103	112	108	106	107	9	5	3	4
4	100	104	102	112	110	4	2	12	10
5	42	44	48	45	45	2	6	3	0
6	82	86	88	102	106	4	6	20	24
7	50	58	52	68	70	8	2	18	20
8	54	60	61	68	70	6	7	14	16
9	110	112	113	117	118	2	3	7	8
10	99	102	106	107	112	3	7	8	13
11	58	59	63	66	70	1	5	8	12
12	80	82	82	86	89	2	2	6	9
13	102	106	105	109	112	4	3	7	10
14	65	63	65	68	72	-2	0	3	7
15	93	98	104	108	108	5	11	15	15
Total	1211	1274	1247	1277	1436	53	70	160	192
Mean	80.73	84.93	83.13	85.13	95.73				

V E C U R O N I U M

S.No.	Blood Glucose level in mg%					II-I	III-I	IV-I	V-I
	I	II	III	IV	V	A	B	C	D
1	108	114	106	120	118	6	-2	12	10
2	94	96	112	106	110	2	8	12	16
3	102	109	105	106	112	7	3	4	10
4	76	92	92	96	94	16	16	20	18
5	74	76	80	90	94	2	6	16	20
6	102	100	108	110	116	-2	6	8	14
7	81	80	94	90	92	-1	13	9	11
8	72	78	78	75	80	6	6	3	8
9	122	120	134	135	130	-2	12	13	8
10	88	90	96	96	100	2	8	8	12
11	103	105	108	113	110	2	5	10	7
12	107	107	110	112	110	0	3	5	3
13	52	62	65	63	66	10	13	11	14
14	40	42	46	52	52	2	6	12	12
15	48	56	53	54	60	8	5	6	12
Total	1225	1327	1387	1418	1444	58	108	149	179
Mean	81.66	88.46	92.46	94.53	96.26				

SUMMARY AND CONCLUSION

SUMMARY

The present study entitled "The study of changes in blood glucose level during general anaesthesia, in patients under going surgery" was carried out at M.L.B. Medical College Hospital Jhansi over a period of one year. The patients were divided into four groups depending upon the use of anaesthetic drugs.

For anaesthetic drugs were used:

1. Ether — Inhalational agent
2. Pancuronium
3. Gallamine — Non depolarising agents
4. Vecuronium

Ether is in use for a long period as a inhalational agent in the form of ether vapour. In the developed countries it is not in use now a days but in India being a devloping country it is still in use.

Non depolarising muscle relaxants revolutionized the anaesthetic practice. These drugs produce active muscular relaxation without unwanted side effects. They have made it possible for the anaesthesiologist to have adequate control over ventilation of the patients.

There is a clinical need for a new non-depolarizing muscle relaxant of shorter duration less cumulative property and fewer side effects. These new muscle relaxant will no doubt changes our pattern of practice by improving the safety and clinical relaxation.

ETHER

It was prepared by Valerius Cordus in 1540 and Sigmund August Forbenisius named it ether and was used in anaesthesia by W.E. Clarke in 1842.

It is volatile liquid of molecular weight 74 and boiling point 35°C specific gravity of vapour being 2.6. Ether vapour is flammable in air. Chemically it is inert and can be stored in dark cool place unaltered in body and 85-90% is eleminated by lungs. This causes increases in heart rate at first due to (i) Catecholamine libration (ii) Sympathatic stimulation and Vagal depression later the heart rate is relatively unchanged.

There are some side effect of ether.

1. Nauses and vomiting post operatively.
2. Explosive risk when used with O_2 and N_2O.
3. Slow induction and recovery from anaesthesia.

PANCURONIUM BROMIDE

Pancuronium bromide is a bisquaternery aminosteroid, described by Buckett, Hewett and Savage in 1967. It appeared to be an effective long acting non-depolarising neuromuscular blocking agent, without the evidence of steroid activity. The paralysis remained between 25 to 45 minuts.

Pancuronium is a markedly cumulative drug. It causes very little hystamine releare and mild transient depression of plasma cholenesterae. Pancuronium is known to have no effect on the liver or any influence over the carbohydrate metabolism causing no significant change in blood sugar level.

GALLAMINE TRIETHIODIDE

Gallamine triethiodide a synthetic nondepolarising muscle relaxant was first described by Bovet et al in 1947 and used in anaesthetic practice by Huguenard et al in 1947.

Chemically Gallamine is tri (B-diethyl amino ethoxy) Benzine tri ethiodide. It is intermediate acting agent with duration of action between 20-30 minutes.

Tachycardia and hypertension occur in the use of this drug but it has no direct or indirect effect over the liver function or carbohydrate metabolism.

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Vecuronium bromide is a recently introduced nondepolarising neuromuscular blocking agent developed by Savage in 1979.

Chemically the drug is the monoquaternary homologue of Pancuronium. However the sterioisometric relation-ship of the 3 acetyle group to the parent ring makes it structurally dis-similar. Advantage of this drug is its short duration of action in low doses and its lack of curmulative effect and its minimal side effect on C.V.S. liver and carbohydrate metabolism in doses upto 20 times that required for paralysis.

Vecuronium with its remarkable lack of effect on C.V.S. liver and other organ, is probably an example of the forerunner to a new generation of neuromuscular blocking agents which provide the anaesthetist with safer and more efficient means of producing muscle relaxation.

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The present study was planned with aim to study the changes in blood glucose level during general anaesthesia in patients under going surgery under ether, pancuronium bromide, gallamine triethiodide and vecuronium bromide.

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CONCLUSION

From the present study we concluded that in comparisation of muscle relaxant ether causes much more hyperglycaemia during general anaesthesia which is very highly significant rise.

But in case of muscle relaxant the change in blood glucose level is just significant.